Brief Report

Oxidative stress and antioxidant markers in patients with alopecia areata: A comparative cross-sectional study

Safoura Shakoei, Hossein Mirmiranpoor[1], Manouchehr Nakhjavani[1], Maryam Nasimi[2], Ghazaleh Bakhshi, Arghavan Azizpour[2]

Department of Dermatology, Imam Khomeini Hospital, [1]Endocrinology and Metabolism Research Center (EMRC), Vali-Asr Hospital, [2]Dermatology, Razi Hospital, Tehran University of Medical Sciences (TUMS), Tehran, Iran.

Abstract

Background: Alopecia areata is a chronic inflammatory skin disease. Oxidative stress may contribute to the pathogenesis of this condition.

Aim: To evaluate the serum oxidative stress markers and antioxidant capacity in patients with alopecia areata.

Methods: This cross-sectional study was performed on 40 patients with alopecia areata and 40 healthy controls. The fasting blood sugar, C-reactive protein, lipid profile, and serum oxidative markers, including advanced glycation end products and advanced oxidation protein products, were measured in this study. Also, antioxidant enzymes, including paraoxonase-1, lecithin-cholesterol acyltransferase and serum ferric-reducing antioxidant power, were determined.

Results: The serum levels of advanced glycation end products and advanced oxidation protein products were significantly higher in patients with alopecia areata, compared to the controls ($P < 0.001$), whereas the levels of ferric-reducing antioxidant power, paraoxonase-1 and lecithin-cholesterol acyltransferase were significantly lower in patients with alopecia areata, compared to the controls ($P < 0.001$). The mean fasting blood sugar level was significantly higher in patients with alopecia areata, compared to the controls. The ferric reducing antioxidant power level was significantly associated with the percentage of hair loss ($P = 0.01$, $r = 0.4$) and the serum C-reactive protein level ($P = 0.03$, $r = –0.3$) in patients with alopecia areata.

Limitations: Since the current study had a cross-sectional design, no cause-effect relationship was established between alopecia areata and oxidative stress. The sample size of our study was also small.

Conclusion: Based on the present results, the oxidant-antioxidant enzymatic system is impaired in alopecia areata due to the increased oxidative products and decreased antioxidant activity.

Key words: Alopecia areata, advanced glycation end product, lecithin-cholesterol acyltransferase, oxidative stress, PON1

Plain Language Summary

Alopecia areata (AA) is a chronic inflammatory disease. Oxidative stress may contribute to the pathogenesis of this condition. So, we evaluated the serum oxidative stress markers and antioxidant capacity in patients with alopecia areata. This cross-sectional study was performed on 40 patients with alopecia areata and 40 healthy controls. Serum oxidative markers, including advanced glycation end products (AGEs) and advanced oxidation protein products (AOPPs), were measured. Paraoxonase-1 (PON1), lecithin-cholesterol acyltransferase (LCAT) and serum ferric-reducing antioxidant power (FRAP) were determined for antioxidant enzymes in our study. The serum levels of AGEs and AOPPs were significantly higher in patients with alopecia areata, compared to the controls ($P < 0.001$), whereas the levels of FRAP, PON1, and LCAT were significantly lower in patients with alopecia areata ($P < 0.001$). According to the results of this study, the oxidant-antioxidant enzymatic system is impaired in alopecia areata due to the increased oxidative products and decreased antioxidant activity.

How to cite this article: Shakoei S, Mirmiranpoor H, Nakhjavani M, Nasimi M, Bakhshi G, Azizpour A. Oxidative stress and antioxidant markers in patients with alopecia areata: A comparative cross-sectional study. Indian J Dermatol Venereol Leprol 2023;89:411-5.

Corresponding author: Dr. Safoura Shakoei, Department of Dermatology, Imam Khomeini Hospital, Tehran University of Medical Sciences (TUMS), Tehran, Iran. dr.shakoei@gmail.com

Received: March, 2020 **Accepted:** February, 2022 **EPub Ahead of Print:** July, 2022 **Published:** April, 2023

DOI: 10.25259/IJDVL_228_20 **PMID:** 35962507

Introduction

Alopecia areata is a common autoimmune skin disorder, with an overall pooled prevalence of 2.11%.[1,2] Infiltration of T-cell lymphocytes around the hair follicles during the anagen phase plays a pivotal role in this disorder.[3] Other triggering factors, including oxidative stress and genetics are also associated with the development of alopecia areata.[4,5] It is known that the skin is continuously exposed to reactive oxygen species, generated by both endogenous and exogenous sources. A balance between oxidative damage and antioxidant protection is necessary for different biological processes.[6] Also, reactive oxygen species plays a key role in apoptosis for reacting with all macromolecules (e.g., lipids, proteins, and carbohydrates) on the cell membrane.[7]

Paraoxonase-1 and lecithin-cholesterol acyltransferase are enzymes with antioxidant activities.[8-10] The ferric reducing antioxidant power assay is used to measure the antioxidant potential of body samples.[11] Also, advanced glycation end products are glycated proteins that lead to the formation of reactive oxygen species through interactions with advanced glycation end product-specific receptors and result in increased oxidative stress.[12] On the other hand, advanced oxidation protein products are oxidized protein products, with a similar structure and biological activity to advanced glycation end products formed during oxidative stress.[13]

The role of products derived from glycosylation, protein oxidation, lecithin-cholesterol acyltransferase and ferric-reducing antioxidant power has been evaluated in a wide range of inflammatory diseases[10,13-15]; but limited studies have been done in alopecia areata patients. Protein and glycation end-products are more stable than lipids and may be better markers of oxidative stress.[12,13] Therefore, in this cross-sectional study, we aimed to investigate and compare the levels of oxidative markers (i.e., advanced oxidation protein product and advanced glycation end product) and antioxidants (i.e., paraoxonase-1, lecithin-cholesterol acyltransferase and ferric-reducing antioxidant power) in patients with alopecia areata and healthy controls.

Methods

This cross-sectional study was performed on 40 patients with alopecia areata who were treated in outpatient dermatology clinics of Razi and Imam Komeini Hospitals during 2018–2019. Forty age-, gender- and body mass index (BMI)-matched healthy individuals with aesthetic complaints were also allocated to the control group. An experienced dermatologist confirmed the diagnosis of alopecia areata. The exclusion criteria were as follows: History of inflammatory or autoimmune diseases other than alopecia areata; infection; diabetes mellitus; familial hyperlipidemia; liver or kidney disorders; malignancies; pregnancy or lactation; consumption of antioxidants, vitamins and topical or systemic anti-inflammatory drugs within the past three months; recent history of major surgery; obesity; alcohol or tobacco use; and, professional athlete.

In agreement with the study conducted by Bilgili *et al.* the sample size was calculated using G*power version 3.1.9.4 ($d = 0.64$, $\alpha = 0.05$ and power = 0.8), with an equal allocation ratio for the two-tailed test and assumption of a normal parent distribution. Finally, 40 samples were calculated for each group.[8] The study was conducted according to the principles outlined in the Declaration of Helsinki and the Medical Research Involving Human Subjects Act.

The clinical information and demographic data of the subjects were documented. The severity of alopecia areata was determined using the Severity of Alopecia Tool.[16] Their BMIs were calculated as weight in kilograms divided by the square of height (kg/m^2). A 10-mL venous blood sample was taken from each participant at around 8–9 AM after a 12-hour fasting period to measure the fasting blood sugar level, C-reactive protein level, lipid profile and other laboratory markers. The lipid profile and fasting blood sugar levels were measured using standard enzymatic-colourimetric methods (Pars Azmun Co. Ltd., Tehran, Iran). The serum C-reactive protein level was assessed using a two-site ELISA assay (Diagnostic Biochem, Ontario, Canada).

Additionally, the levels of the advanced glycation end product and advanced oxidation protein product were measured, using the spectrophotometric method (FLUOstar OPTIMA reader, BMG, Germany).[13] The serum paraoxonase-1 level was also measured, using an automated paraoxonase assay kit (V31137, ZellBio Co., Germany), following a colourimetric method. Also, to determine the lecithin-cholesterol acyltransferase activity, a commercially available kit (Calbiochem Co., USA) was used, based on the fluorometric method, and the results were expressed as the ratio of wavelengths (470 nm/390 nm). Finally, ferric-reducing antioxidant power was measured using spectrophotometry.[11]

Data analysis was performed using SPSS version 24 (Chicago, IL, USA). For group comparisons, independent sample *t*-test, Mann-Whitney *U* test and Chi-square tests were used. Linear regression analysis was also performed to determine the relationships between continuous variables. A *P*-value of less than 0.05 was considered statistically significant.

Results

This study was conducted on 40 patients with alopecia areata and 40 healthy controls, including 21 (52.5%) females and 19

Table 1: Baseline demographics, clinical characteristics and laboratory findings of patients with alopecia areata and healthy controls

Characteristic	Patients with alopecia areata (*n* = 40)	Healthy controls (*n* = 40)	*P*-value*
Gender, *n* (%)			1
Female	21	21	
Male	19	19	
Age, years	29.22 ± 10.69	29.32 ± 11.81	0.96
BMI	25.07 ± 4.183	26.21 ± 7.15	0.38
Duration of current disease, months	36.71 ± 66.69	–	
Onset of disease, years	18.4 ± 10.90	–	
Type of alopecia areata, No. (%)			
Patchy	55	–	
Ophiasis	0	–	
Totalis	7.5	–	
Universalis	37.5	–	
Nail involvement, *n* (%)	62.5	–	
FBS	100.87 ± 15.03	92.82 ± 11.62	**<0.001**
Total cholesterol, mg/dL	162.50 ± 43.06	172.35 ± 33.26	0.25
LDL, mg/dL	103.27 ± 33.71	107.83 ± 23.80	0.48
HDL, mg/dL	36.62 ± 8.63	39.75 ± 8.85	0.11
TG, mg/dL	113 ± 59.93	123.85 ± 82.49	0.50
CRP	2.78 ± 3.55	2.96 ± 3.04	0.80
AGE (µmol/L)	78.25 ± 4.44	46.48 ± 4.75	**<0.001**
AOPP (µmol/L)	182.82 ± 5.99	106.88 ± 7.99	**<0.001**
FRAP (µmol/L)	822.50 ± 49.43	1400 ± 165.74	**<0.001**
PON1 (U/l)	96.96 ± 10.90	206.82 ± 8.62	**<0.001**
LCAT (nmol/ml/hr.)	34.87 ± 3.91	55.46 ± 3.07	**<0.001**

N: number, BMI: body mass index, FBS: fasting blood sugar, LDL: low-density lipoprotein, HDL: high-density lipoprotein, TG: triglyceride, CRP: c-reactive protein, AGE: advanced glycation end product, AOPP: advanced oxidation protein product, FRAP: ferric-reducing antioxidant power, PON1: paraoxonase 1, LCAT: lecithin cholesterol acyltransferase, *a *P*-value of less than 0.05 is considered significant

Table 2: Correlations between serum markers and risk factors in AA patients

Source	AGES *	AOPP *	FRAP *	PON1 *	LCAT *
Age	0.50(-0.11)	0.18(0.21)	0.34(-0.15)	0.42(-0.12)	0.11(-0.25)
Onset age of disease	0.41(0.13)	0.70(0.06)	0.14(-0.23)	0.94(-0.01)	0.98(-0.004)
SALT score	0.7(-0.06)	0.83(-0.03)	**0.01(0.4)**	0.87(0.02)	0.97(0.004)
FBS	0.57(-0.09)	0.34(0.15)	0.05(0.31)	0.77(-0.04)	0.26(0.17)
Cholesterol	**0.04(-0.03)**	0.07(0.28)	0.46(0.12)	0.23(-0.19)	0.17(-0.21)
TG	0.53(-0.1)	0.21(0.2)	0.29(0.16)	**0.02(-0.35)**	0.92(0.01)
HDL	0.4(-0.13)	0.41(0.13)	0.34(-0.15)	0.48(0.03)	0.21(-0.12)
LDL	0.1(-0.25)	0.07(0.28)	0.34(0.15)	0.48(-0.11)	0.21(-0.2)
CRP	0.34(0.15)	0.44(-0.12)	**0.03(-0.33)**	0.5(0.1)	0.62(0.08)

**P*-value (Correlation Coefficient, CI), *P*-value of less than 0.05 is considered significant. AGEs: Advanced glycation end products, AOPP: Advanced oxidation protein products, FRAP: Ferritin reducing the ability of plasma, PON: Paraoxonase1, LCAT: Lecithin cholesterol acyltransferase, SALT score: the Severity of Alopecia Tool, FBS: fasting blood sugar, LDL: low-density lipoprotein, HDL: high-density lipoprotein, TG: triglyceride, CRP: c-reactive protein

Table 3: Evaluation of the relationship between involvement pattern and oxidative and antioxidative markers in patients group

	Mean ± SD			*P*-value
	Patchy	Totalis	Universalis	
AGES	78.77 ± 4.18	75 ± 5.19	78.13 ± 4.71	0.39
AOPP	183.34 ± 6.05	191.99 ± 6.52	182.82 ± 5.99	0.8
FRAP	808.55 ± 51.82	855 ± 67.43	836.47 ± 37.25	0.11
PON1	96.92 ± 10.93	95.87 ± 6.53	97.23 ± 12.07	0.98
LCAT	35.04 ± 3.69	34.43 ± 4.54	34.72 ± 4.38	0.95

P-value of less than 0.05 is considered significant. AGEs: advanced glycation end products, AOPP: advanced oxidation protein products, FRAP: ferritin reducing the ability of plasma, PON: paraoxonase1, LCAT: lecithin cholesterol acyltransferase

(47.5%) males in each group. The mean age of the subjects was 29.23 ± 10.69 years in the alopecia areata group and 29.32 ± 11.81 years in the control group ($P = 0.96$). Other baseline demographics, clinical characteristics and laboratory findings of the patients and healthy controls are presented in Table 1.

The mean serum levels of advanced glycation end products and advanced oxidation protein product were significantly higher in patients with alopecia areata (78.25 ± 4.44 and 182.82 ± 5.99, respectively), compared to the controls (46.48 ± 4.75 and 106.88 ± 7.99, respectively) ($P < 0.001$). In contrast, the mean serum levels of antioxidant markers, including ferric-reducing antioxidant power, paraoxonase-1 and lecithin-cholesterol acyltransferase, were significantly lower in the alopecia areata group (822.50 ± 49.43, 96.96 ± 10.90 and 34.87 ± 3.91, respectively), compared to the controls (1400 ± 165.74, 206.82 ± 8.62 and 55.46 ± 3.07, respectively) ($P < 0.001$) [Table 1]. The results showed that the mean fasting blood sugar level was significantly higher in the alopecia areata group, compared to the controls (100.87 ± 15.03 vs. 92.82 ± 11.62; $P < 0.001$).

There was a significant difference in the advanced glycation end product level between female and male patients with alopecia areata (79.9 ± 3.8 in females vs. 76.42 ± 4.5 in males; $P = 0.01$). A negative correlation was found between the serum advanced glycation end product and cholesterol levels in the alopecia areata group ($r = –0.03$, $P = 0.04$). Statistical analysis based on Spearman's rho test on the alopecia areata group revealed a positive correlation between the ferric-reducing antioxidant power level and the percentage of hair loss ($P = 0.01$, $r = 0.4$), and a negative correlation between ferric-reducing antioxidant power level and C-reactive protein ($P = 0.03$, $r = –0.3$). Moreover, the serum triglyceride level was negatively correlated with the paraoxonase-1 activity in this group ($P = 0.02$, $r = –0.35$) [Table 2]. The oxidative

and antioxidant markers had no significant associations with hair-loss patterns ($P > 0.05$) [Table 3].

Discussion

The present results revealed that the mean serum levels of markers of oxidative stress were significantly higher in patients with alopecia areata, compared to the controls, whereas the mean antioxidant activity was significantly lower in the alopecia areata group, compared to the controls. The SALT score and the hair-loss pattern had no significant associations with oxidative and antioxidant markers. Moreover, we found a negative correlation between C-reactive protein and ferric-reducing antioxidant power levels. C-reactive protein (CRP) is an inflammatory mediator and was significantly increased in alopecia areata patients.[17] Although our study did not support this result, the possible role of oxidative stress and decreased antioxidative markers like ferric-reducing antioxidant power with inflammatory biomarkers in the development of alopecia areata. CRP level was not higher in alopecia areata cases. But the possible role of oxidative stress and decreased antioxidative markers like FRAP with inflammatory biomarkers in the development of alopecia areata were indicated.

Generally, it is suggested that oxidative stress plays a critical role in the pathogenesis of autoimmune disorders, including psoriasis,[18] vitiligo[19] and pemphigus vulgaris,[20] by causing inflammation, inducing cell apoptosis, and reducing immune tolerance. Although some studies have supported the role of oxidative stress in the pathogenesis of alopecia areata,[21,22] the results are inadequate and conflicting.[23,24]

There are also reports of increased lipid peroxidation and decreased activities of antioxidants, such as glutathione, glutathione peroxidase, and superoxide dismutase, in patients with alopecia areata, compared to the controls.[25] In this regard, Barky and Bilgili *et al.* found that the mean total antioxidant capacity in the serum was significantly lower in patients with alopecia areata, compared to the controls.[8,22] Our results also showed that the antioxidant activity was significantly lower in patients with alopecia areata compared to healthy controls. On the other hand, Akar *et al.* found an increase in the antioxidant activity of superoxide dismutase and glutathione peroxidase in the scalp of patients with alopecia areata, especially in the early stages of the disease, whereas no reduction was observed in the lipid peroxidation rate.[24] The results of an Akar study were contrary to our study.

Evidence suggests that paraoxonase-1 is significantly reduced in patients with alopecia areata. This enzyme is majorly influenced by gender, as its activity is higher in females than males.[26] However, the present study showed that paraoxonase-1 decrease in patients with alopecia areata but did not confirm any link between paraoxonase-1 activity and gender in alopecia areata patients. It is known that advanced oxidation protein products and advanced glycation end products activate the membrane receptors for the latter to trigger reactive oxygen species generation and oxidative stress, which is involved in inflammatory diseases.[13,14] However, most previous studies have addressed the lipid peroxidation rate as a marker of oxidative stress in alopecia areata.[24,27,28] In this regard, Cwynar *et al.* reported a statistically non-significant higher plasma level of advanced oxidation protein products in patients with alopecia areata compared to the controls; however.[29] However, this difference in our study was statistically significant.

The reduced serum levels of lecithin-cholesterol acyltransferase and ferric-reducing antioxidant power have been reported in a wide range of inflammatory diseases, including psoriasis and chronic kidney disease.[18,30,31] Our results also showed that the serum levels of ferric-reducing antioxidant power and lecithin-cholesterol acyltransferase were significantly lower in patients with alopecia areata, compared to the healthy controls.

Several studies have shown that oxidative stress is involved in the pathogenesis of psoriasis.[32] The total oxidant status and malondialdehyde[4] levels were significantly higher in psoriasis patients, whereas total antioxidant status and catalase levels in these patients were significantly lower than in healthy controls.[33] Based on our study results, the imbalance of oxidative and antioxidant activity may be involved in the pathogenesis of alopecia areata, as it is in psoriasis.

This study had some limitations. First, the sample size was small. Second, most correlations, although significant, were weak and difficult to generalise. Therefore, future research with a larger sample size is necessary to confirm our findings.

We were unable to find any previous reports about serum ferric-reducing antioxidant power, lecithin-cholesterol acyltransferase and advanced glycation end-product levels in patients with alopecia areata. Our results indicated an imbalance of the oxidant-antioxidant enzymatic system in alopecia areata. Therefore, the administration of agents with antioxidant activities may be useful in the management of alopecia areata.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

The research is supported by Tehran University of Medical Science.

Conflicts of interest

There are no conflicts of interest.

References

1. Cervantes J, Fertig RM, Maddy A, Tosti A. Alopecia areata of the beard: A review of the literature. Am J Clin Dermatol 2017;18:789–96.

2. Lee SM, Kim MS, Kim YJ, Won CH, Lee MW, Choi JH, *et al.* Adverse events of non-ablative fractional laser photothermolysis: A retrospective study of 856 treatments in 362 patients. J Dermatolog Treat 2014;25:304–07.
3. Cwynar A, Olszewska-Slonina D, Czajkowski R, Zegarska B, Bialecka A, Mecinska-Jundzill K, *et al*. Investigation of oxidative stress in patients with alopecia areata by measuring the levels of malondialdehyde and ceruloplasmin in the blood. Postepy Dermatol Alergol 2018;35:572–76.
4. Dizen-Namdar N, Emel Kocak F, Kidir M, Sarici G, Tak H, Altuntas I. Evaluation of serum paraoxonase, arylesterase, prolidase activities and oxidative stress in patients with alopecia areata. Skin Pharmacol Physiol 2019;32:59–4.
5. Yenin JZ, Serarslan G, Yonden Z, Ulutas KT. Investigation of oxidative stress in patients with alopecia areata and its relationship with disease severity, duration, recurrence and pattern. Clin Exp Dermatol 2015;40:617–21.
6. Shah AA, Sinha AA. Oxidative stress and autoimmune skin disease. Eur J Dermatol 2013;23:5–13.
7. Kannan K, Jain SK. Oxidative stress and apoptosis. Pathophysiology 2000;7:153–63.
8. Bilgili SG, Ozkol H, Karadag AS, Ozkol HU, Seker A, Calka O, *et al.* Serum paraoxonase activity and oxidative status in subjects with alopecia areata. Cutan Ocul Toxicol 2013;32:290–93.
9. Sigales TS, Uliano G, Muniz L, Barros C, Schneider A, Valle SC. Influence of nutritional factors and the PON1 C(-107)T polymorphism on paraoxonase-1 activity in childhood. Jornal de pediatria 2020;96:495–502.
10. Hansel B, Kontush A, Bonnefont-Rousselot D, Bruckert E, Chapman MJ. Alterations in lipoprotein defense against oxidative stress in metabolic syndrome. Curr Atheroscler Rep 2006;8:501–9.
11. Benzie IF, Strain JJ. The ferric reducing ability of plasma (FRAP) as a measure of "antioxidant power": The FRAP assay. Anal Biochem 1996;239:70–6.
12. Cho S-J, Roman G, Yeboah F, Konishi Y. The road to advanced glycation end products: A mechanistic perspective. Curr Med Chem 2007;14:1653–71.
13. Kalousova M, Skrha J, Zima T. Advanced glycation end-products and advanced oxidation protein products in patients with diabetes mellitus. Physiol Res 2002;51:597–604.
14. Papagrigoraki A, Maurelli M, Del Giglio M, Gisondi P, Girolomoni G. Advanced glycation end products in the pathogenesis of psoriasis. Int J Mol Sci 2017;18:2471.
15. Ozgor O, Akoglu G, Sungu N, Karaismailoglu E, Aktas A. Receptor for advanced glycation end products is overexpressed in psoriatic plaques independent of disease severity. J Dermatol Venereol Leprol 2017;83:556–60.
16. Olsen EA, Hordinsky MK, Price VH, Roberts JL, Shapiro J, Canfield D, *et al.* Alopecia areata investigational assessment guidelines–Part II. National Alopecia Areata Foundation. J Am Acad Dermatol 2004;51:440–47.
17. Mustafa AI, Khashaba RA, Fawzy E, Baghdady SMA, Rezk SM. Cross talk between oxidative stress and inflammation in alopecia areata. J Cosmet Dermatol 2021;20:2305–10.
18. Asha K, Singal A, Sharma SB, Arora VK, Aggarwal A. Dyslipidaemia & oxidative stress in patients of psoriasis: Emerging cardiovascular risk factors. Indian J Med Res 2017;146:708–13.
19. Speeckaert R, Dugardin J, Lambert J, Lapeere H, Verhaeghe E, Speeckaert MM, *et al.* Critical appraisal of the oxidative stress pathway in vitiligo: A systematic review and meta-analysis. J Eur Acad Dermatol Venereol 2018;32:1089–98.
20. Yesilova Y, Ucmak D, Selek S, Dertlioglu SB, Sula B, Bozkus F, *et al.* Oxidative stress index may play a key role in patients with pemphigus vulgaris. J Eur Acad Dermatol Venereol 2013;27:465–67.
21. Ramadan R, Tawdy A, Abdel Hay R, Rashed L, Tawfik D. The antioxidant role of paraoxonase 1 and vitamin E in three autoimmune diseases. Skin Pharmacol Physiol 2013;26:2–7.
22. Bakry OA, Elshazly RM, Shoeib MA, Gooda A. Oxidative stress in alopecia areata: A case-control study. Am J Clin Dermatol 2014;15: 57–64.
23. Motor S, Ozturk S, O zcan O, Gurpinar AB, Can Y, Yuksel R, *et al.* Evaluation of total antioxidant status, total oxidant status and oxidative stress index in patients with alopecia areata. Int J Clin Exp Med 2014;7:1089–93.
24. Akar A, Arca E, Erbil H, Akay C, Sayal A, Gur AR. Antioxidant enzymes and lipid peroxidation in the scalp of patients with alopecia areata. J Dermatol Sci 2002;29:85–90.
25. Naziroglu M, Kokcam I. Antioxidants and lipid peroxidation status in the blood of patients with alopecia. Cell Biochem Funct 2000;18:169–73.
26. Trentini A, Bellini T, Bonaccorsi G, Cavicchio C, Hanau S, Passaro A, *et al.* Sex difference: An important issue to consider in epidemiological and clinical studies dealing with serum paraoxonase-1. J Clin Biochem Nutr 2019;64:250–56.
27. Abdel Fattah NS, Ebrahim AA, El Okda ES. Lipid peroxidation/ antioxidant activity in patients with alopecia areata. J Eur Acad Dermatol Venereol 2011;25:403–8.
28. Güngör Ş, AkbayG, Öğüş E, Ekşioğlu M, Yücel D. Changes of lipid peroxidation and antioxidant system in serum and tissues of patients with alopecia areata. Turkiye Klinikleri Journal of Dermatology 2008;18:141–5.
29. Cwynar A, Olszewska-Slonina D, Czajkowski R, Zegarska B, Wybranowski T, Holynska-Iwan I, *et al.* Evaluation of selected parameters of oxidative stress in patients with alopecia areata. Postepy Dermatol Alergol 2019;36:115–6.
30. Holzer M, Wolf P, Inzinger M, Trieb M, Curcic S, Pasterk L, *et al.* Anti-psoriatic therapy recovers high-density lipoprotein composition and function. J Invest Dermatol 2014;134:635–42.
31. Calabresi L, Simonelli S, Conca P, Busnach G, Cabibbe M, Gesualdo L, *et al.* Acquired lecithin: cholesterol acyltransferase deficiency as a major factor in lowering plasma HDL levels in chronic kidney disease. J Intern Med 2015;277:552–61.
32. Zhou Q, Mrowietz U, Rostami-Yazdi M. Oxidative stress in the pathogenesis of psoriasis. Free Radic Biol Med 2009;47:891–905.
33. Zhang Y, Li Z, Ma Y, Mu Z. Association of total oxidant status, total antioxidant status, and malondialdehyde and catalase levels with psoriasis: A systematic review and meta-analysis. Clin Rheumatol 2019;38:2659–71.